**What is claimed is:**

1. A method for inhibiting fusion of HIV-1 to $CD4^+$ cells which comprises contacting $CD4^+$ cells with a non-chemokine agent capable of binding to a chemokine receptor in an amount and under conditions such that fusion of HIV-1 to the $CD4^+$ cells is inhibited.

2. A method for inhibiting HIV-1 infection of $CD4^+$ cells which comprises contacting $CD4^+$ cells with a non-chemokine agent capable of binding to a chemokine receptor in an amount and under conditions such that fusion of HIV-1 to the $CD4^+$ cells is inhibited, thereby inhibiting HIV-1 infection.

3. The method of claim 1 or 2, wherein the non-chemokine agent is an oligopeptide.

4. The method of claim 1 or 2, wherein the non-chemokine agent is a polypeptide.

5. The method of claim 1 or 2, wherein the non-chemokine agent is an antibody or a portion of an antibody.

6. The method of claim 1 or 2, wherein the non-chemokine agent is a nonpeptidyl agent.

7. A non-chemokine agent capable of binding to a chemokine receptor and inhibiting fusion of HIV-1 to $CD4^+$ cells.

8. The non-chemokine agent of claim 7, wherein the non-chemokine agent is a oligopeptide.

9. The non-chemokine agent of claim 7, wherein the non-chemokine agent is a nonpeptidyl agent.

10. The non-chemokine agent of claim 7, wherein the non-chemokine agent is a polypeptide.

11. The non-chemokine agent of claim 10, wherein the polypeptide is an antibody or a portion of an antibody.

12. The non-chemokine agent of claim 10, wherein the polypeptide comprises amino acid sequence as set forth in SEQ ID NO:5.

13. The non-chemokine agent of claim 10, wherein the polypeptide comprises the MIP-1ß sequence with the deletion of the first seven N-terminal amino acids of said sequence.

14. The non-chemokine agent of claim 10, wherein the polypeptide comprises the MIP-1ß sequence with the deletion of the first eight N-terminal amino acids of said sequence.

15. The non-chemokine agent of claim 10, wherein the polypeptide comprises the MIP-1ß sequence with the deletion of the first nine N-terminal amino acids of said sequence.

16. The non-chemokine agent of claim 10, wherein the polypeptide comprises the MIP-1ß sequence with the deletion of the first ten N-terminal amino acids of said sequence.

17. The non-chemokine agent of claim 10, wherein the polypeptide comprises the MIP-1ß sequence with the N-terminal sequence modified by addition of an amino acid or oligopeptide.

18. The non-chemokine agent of claim 10, wherein the polypeptide comprises the MIP-1ß sequence with the N-terminal sequence modified by removing the N-terminal alanine and replacing it by serine or threonine and an additional amino acid or oligopeptide or nonpeptidyl moiety.

19. The non-chemokine agent of claim 17 or 18, wherein the additional amino acid is methionine.

20. An agent capable of binding to CXCR4 and inhibiting HIV-1 infection.

21. The agent of claim 20, wherein the agent is an oligopeptide.

22. The agent of claim 20, wherein the agent is a polypeptide.

23. The non-chemokine agent of claim 22, wherein the polypeptide comprises the SDF-1 sequence with the deletion of the first six N-terminal amino acids of said sequence.

24. The non-chemokine agent of claim 22, wherein the polypeptide comprises the SDF-1 sequence with the deletion of the first seven N-terminal amino acids of said sequence.

25. The non-chemokine agent of claim 22, wherein the polypeptide comprises the SDF-1 sequence with the deletion of the first eight N-terminal amino acids of said sequence.

26. The non-chemokine agent of claim 22, wherein the

polypeptide comprises the SDF-1 sequence with the deletion of the first nine N-terminal amino acids of said sequence.

27. The non-chemokine agent of claim 22, wherein the N-terminal glycine of SDF-1 is replaced by serine and derivatized with biotin.

28. The non-chemokine agent of claim 22, wherein the N-terminal glycine of SDF-1 is replaced by serine and derivatized with methionine.

29. The non-chemokine agent of claim 22, wherein the N-terminus of SDF-1 is modified by the addition of a methionine before the terminal glycine.

30. The agent of claim 22, wherein the agent is an antibody or a portion of an antibody.

31. The agent of claim 20, wherein the agent is a non-peptidyl agent.

32. A pharmaceutical composition comprising an amount of the non-chemokine agent of claim 7 effective to inhibit fusion of HIV-1 to $CD4^+$ cells and a pharmaceutically acceptable carrier.

33. A pharmaceutical composition comprising an amount of the non-chemokine agent of claim 20 effective to inhibit fusion of HIV-1 to $CD4^+$ cells and a pharmaceutically acceptable carrier.

34. A composition of matter capable of binding to a chemokine receptor and inhibiting fusion of HIV-1 to $CD4^+$ cells comprising a non-chemokine agent linked to

a ligand capable of binding to a cell surface receptor of the $CD4^+$ cells other than the chemokine receptor such that the binding of the non-chemokine agent to the chemokine receptor does not inhibit the binding of the ligand to the other receptor.

35. The composition of matter of claim 34, wherein the cell surface receptor is CD4.

36. The composition of matter of claim 34, wherein the ligand comprises an antibody or a portion of an antibody.

37. A pharmaceutical composition comprising an amount of the composition of matter of claim 34 effective to inhibit fusion of HIV-1 to $CD4^+$ cells and a pharmaceutically acceptable carrier.

38. A composition of matter capable of binding to the chemokine receptor and inhibiting fusion of HIV-1 to $CD4^+$ cells comprising a non-chemokine agent linked to a compound capable of increasing the *in vivo* half-life of the non-chemokine agent.

39. The composition of matter of claim 38, wherein the compound is polyethylene glycol.

40. A pharmaceutical composition comprising an amount of the composition of claim 38 effective to inhibit fusion of HIV-1 to $CD4^+$ cells and a pharmaceutically acceptable carrier.

41. A method for reducing the likelihood of HIV-1 infection in a subject comprising administering the pharmaceutical composition of claim 32, 33, 37 or 40 to

the subject.

42. A method for treating HIV-1 infection in a subject comprising administering the pharmaceutical composition of claim 32, 33, 39 or 40 to the subject.

43. A method for determining whether a non-chemokine agent is capable of inhibiting the fusion of HIV-1 to a $CD4^+$ cell which comprises:

(a) contacting (i) a $CD4^+$ cell, which is labeled with a first dye, with (ii) a cell expressing the HIV-1 envelope glycoprotein on its surface, which is labeled with a second dye, in the presence of an excess of the agent under conditions permitting the fusion of the $CD4^+$ cell to the cell expressing the HIV-1 envelope glycoprotein on its surface in the absence of the agent, the first and second dyes being selected so as to allow resonance energy transfer between the dyes;

(b) exposing the product of step (a) to conditions which would result in resonance energy transfer if fusion has occurred; and

(c) determining whether there is a reduction of resonance energy transfer, when compared with the resonance energy transfer in the absence of the agent, a decrease in transfer indicating that the agent is capable of inhibiting fusion of HIV-1 to $CD4^+$ cells.

44. The method of claim 43, wherein the agent is an oligopeptide.

45. The method of claim 43, wherein the agent is a polypeptide.

46. The method of claim 43, wherein the agent is an antibody or a portion of an antibody.

47. The method of claim 43, wherein the agent is a nonpeptidyl agent.

48. The method of claim 43, wherein the $CD4^+$ cell is a PM1 cell.

49. The method of claim 43, wherein the cell expressing the HIV-1 envelope glycoprotein is a HeLa cell expressing $HIV\text{-}1_{JR\text{-}FL}$ gp120/gp41.

50. The method of claim 43, wherein the cell expressing the HIV-1 envelope glycoprotein is a HeLa cell expressing $HIV\text{-}1_{LAI}$ gp120/gp41.